

REPLY:

We thank Wostyn et al for their interest in our recent article. We initially hypothesized that astronauts presenting with the spaceflight-associated neuro-ocular syndrome (SANS) would have ventricular enlargement at postflight MR imaging. Instead, we found greater percentage changes in pre- to postflight ventricular size in the astronauts without SANS. In this regard, we find the comment of Wostyn et al very interesting. The authors suggest that “microgravity-induced ocular changes may be partially determined by the elastic properties of the brain ventricles and optic nerve sheath” and that the “CSF compartment... may act as buffer zones against the development of SANS.” In fact, we have also previously hypothesized that viscoelastic properties may contribute to the development of ventricular enlargement, which may represent a neuroadaptive response to spaceflight.^{1,2} We have made recommendations to the National Aeronautics and Space Administration (NASA) to consider the inclusion of MR elastography as a noninvasive imaging measure of brain stiffness,³ which may serve as a preflight predictor of who may be more susceptible to the development of SANS. Most important, we believe that NASA should consider the inclusion of advanced MR imaging protocols, such as MR elastography, to further explore the mechanisms underlying both the ophthalmologic changes of SANS and the structural changes to the brain experienced by astronauts on long-term missions to the International Space Station. This is particularly critical as we plan for future missions to the Moon or Mars.

We also believe that investigating the response of the intracranial compartment to the spaceflight environment may provide new insights into the understanding of CSF disorders here on Earth.² For example, it could be that patients who develop idiopathic intracranial hypertension (IIH) have less compliant brains without the ability of the ventricular system to enlarge in times of

elevated CSF pressures. As Wostyn et al have proposed for astronauts with SANS, perhaps, too, in patients with IIH, the ventricles may not act as a buffer, leading to the development of optic disc edema. This hypothesis is supported by the characteristic finding in patients with IIH of slit-like ventricles on MR imaging. We look forward to further studies of intracranial physiology during spaceflight both to ensure the safety of our space explorers and to benefit patients with disorders of CSF homeostasis on Earth.

REFERENCES

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 **D.R. Roberts**

 **D. Asemani**


Department of Radiology and Radiological Science

 **P.J. Nietert**

Department of Public Health Sciences

 **M.A. Eckert**


Department of Otolaryngology–Head and Neck Surgery

 **D.C. Inglesby**

Department of Radiology and Radiological Science

Medical University of South Carolina


Charleston, South Carolina

 **J.J. Bloomberg**

Neurosciences Laboratory

NASA Johnson Space Center

Houston, Texas


 **M.S. George**

Department of Psychiatry and Behavioral Sciences

Medical University of South Carolina

Ralph H. Johnson VA Medical Center

Charleston, South Carolina

 **T.R. Brown**

Department of Radiology and Radiological Science

Medical University of South Carolina

Charleston, South Carolina

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